

Cardiac Tamponade by Loculated Pericardial Hematoma: Limitations of M-Mode Echocardiography

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Three patients developed cardiac tamponade after heart surgery. Pericardial effusion was not identified by M-mode echocardiography. Two patients underwent two-dimensional echocardiography which showed compression of the right atrium by a localized mass. At reoperation, atrial compression by a loculated effusion or he-

matoma was found and successfully relieved. When cardiac tamponade is suggested by the clinical setting, but not supported by M-mode echocardiography, the presence of a loculated effusion should be considered and evaluated by a two-dimensional echocardiographic study.

Cardiac tamponade is a life-threatening condition that is curable. Its presence is suggested by signs of a decreased cardiac output, distended neck veins and frequently a paradoxical pulse. In this clinical setting, echocardiography has become the standard tool for diagnosis of pericardial effusion, which is usually the anatomic basis for cardiac tamponade. In many instances, M-mode echocardiography is available at the bedside, and is sufficient to establish the diagnosis of pericardial effusion and even to suggest signs of cardiac tamponade. However, this must not lead to the conclusion that detection of effusion by M-mode echocardiography is a requisite to the diagnosis of cardiac tamponade.

We describe three patients who developed cardiac tamponade after open heart surgery. In each patient, M-mode echocardiography did not demonstrate pericardial effusion. In two patients, two-dimensional echocardiography showed compression of the right atrium. Surgical exploration in all three patients revealed a large pericardial hematoma that compressed the right atrium. Removal of the hematoma eliminated hemodynamic abnormalities.

Case Reports

Case 1. A 62 year old man with severe angina pectoris was referred for coronary bypass surgery. He had triple saphenous vein aortocoronary bypass grafting. Eighteen hours later, he became hypotensive; the systolic blood pressure was 80 mm Hg and did

not vary with respiration. Elevation of the right atrial pressure to 22 cm H₂O was noted, and the pulmonary capillary wedge pressure was 16 mm Hg. The cardiac output decreased from 5.0 to 3.2 liters/min. The urinary output decreased from 60 to 10 cc/h. M-mode echocardiography showed normal-sized chambers and normal valve motion. There was no significant respiratory variation in chamber size and no evidence of pericardial effusion.

Two-dimensional echocardiography was then performed, using the standard techniques (1). The long axis view revealed that ventricles, aortic root, left atrium and valves were normal. The short axis view also appeared normal, but the right atrium was not well delineated. However, in the four chamber view (Fig. 1), a large, 5 × 5 cm mass appeared to compress the right atrium from the right side. The atrium was reduced to a slit-like chamber. The left atrium and both ventricles appeared to be of normal size, and there was no other evidence of pericardial effusion. These findings were interpreted as a localized right atrial compression, probably by hematoma or clot, and the patient was taken to the operating room for exploration.

A thoracotomy was performed and the pericardium was opened. A small amount of bloody pericardial effusion was noted anteriorly. There was, however, a large, loculated hematoma consisting of fresh and clotted blood that compressed the right atrium at its junction with the superior vena cava. The hematoma was evacuated and the pericardium was closed. Immediately, blood pressure rose to 120/75 mm Hg, right atrial pressure decreased to 8 cm H₂O and urinary output increased. Echocardiography was repeated and showed that the evidence of right atrial compression was no longer present.

Case 2. A 38 year old man underwent mitral valve replacement for rheumatic mitral stenosis and insufficiency. The first 3 postoperative days were uneventful and anticoagulation with sodium warfarin (Coumadin) was started. On the 5th postoperative day the patient complained of shortness of breath. The electrocardiogram was unchanged. The chest X-ray film showed cardiomegaly,

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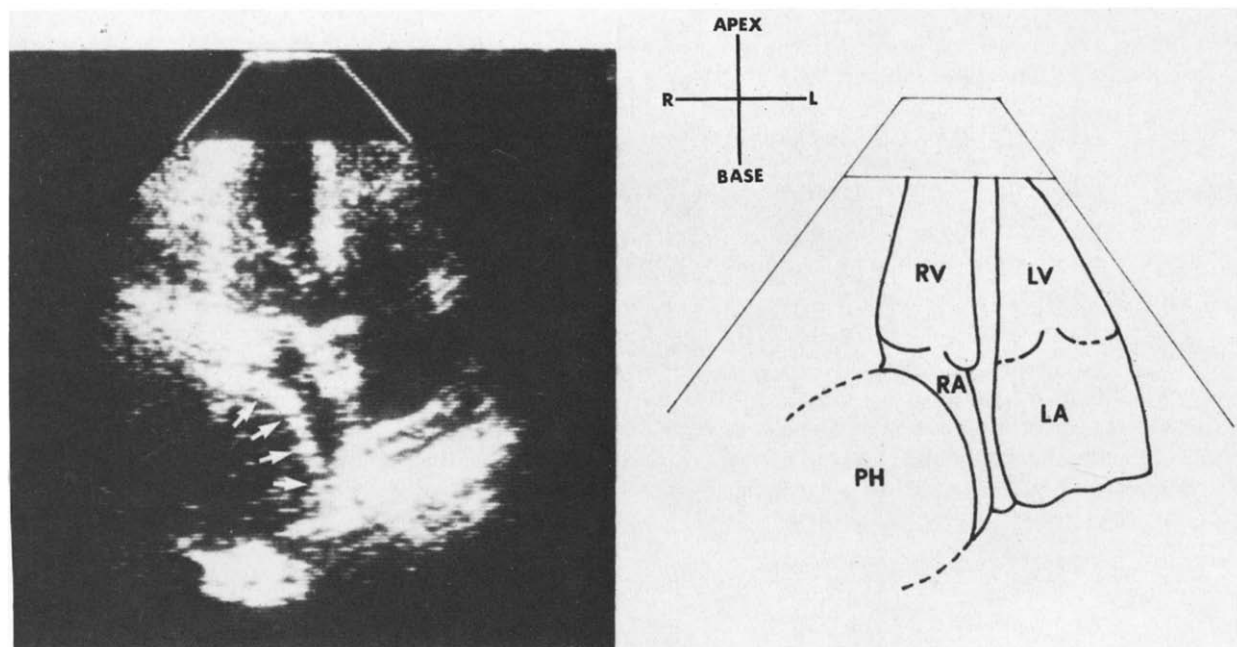


Figure 1. Apical four chamber view of two-dimensional echocardiogram showing a large pericardial hematoma (PH) compressing the right side of the right atrial wall (arrows). Note the slit-like appearance of the right atrial cavity (RA). L = left; LA = left atrium, LV = left ventricle, R = right; RV = right ventricle.

a prosthetic Edwards-Carpentier mitral valve and no evidence of pulmonary venous congestion.

M-mode echocardiography showed left atrial dilation, slight left ventricular dilation, paradoxical motion of the interventricular septum and a prosthetic porcine mitral valve. There was no echo-free space in front of or behind the heart. On the 6th postoperative day the patient became hypotensive. The blood pressure was 70/40 mm Hg, and neck vein distension was noted. Right-sided catheterization with a Swan-Ganz catheter revealed an elevated right ventricular diastolic pressure (18 mm Hg) and an elevated mean right atrial pressure (18 mm Hg). Anuria developed and the patient became confused.

He was immediately taken to the operating room for exploration. A large loculated pericardial hematoma was noted in the oblique sinus of the pericardial sac, producing right and left atrial compression. The hematoma was evacuated and immediately right atrial pressure and urinary output returned to normal. The rest of the hospital course was unremarkable.

Case 3. A 50 year old woman underwent aortic valve replacement. Anticoagulant therapy was started on the 4th postoperative day. The patient was discharged from the hospital without any complications on the 10th postoperative day. One week later, she complained of shortness of breath and was brought to the emergency room. On admission she was diaphoretic. The blood pressure was 60 mm Hg and marked neck vein distension was noted. An inspiratory decrease of 10 mm Hg in the systolic blood pressure was also observed. The electrocardiogram showed left ventricular

hypertrophy and was not changed from the discharge electrocardiogram.

M-mode echocardiography showed a prosthetic aortic valve, mild left atrial dilation (4.5 cm), concentric left ventricular hypertrophy and no sign of pericardial effusion. Two-dimensional echocardiography in the four chamber view showed compression of the right atrium by a large mass. In view of the clinical picture, the study was interpreted as consistent with isolated compression of the right atrium by clot or hematoma.

The patient was immediately transferred to the operating room for exploration of the pericardium. There was no pericardial effusion around the ventricles. However, a large, loculated hematoma compressed the right atrium from the right side. This hematoma was evacuated and immediately blood pressure returned to normal and neck vein distension disappeared. Right atrial pressure decreased from 22 to 4 mm Hg.

Discussion

Echocardiographic signs of cardiac tamponade. Cardiac tamponade is usually associated with pericardial effusion. The criteria for echocardiographic identification of cardiac tamponade include: 1) the demonstration of significant pericardial effusion, and 2) signs of cardiac compression. Pericardial effusion is usually recognized as an echo-free space in front of the right ventricle and behind the left ventricle. Usually, no pericardial effusion is seen behind the left atrium (2,3). Signs of compression include decrease of right ventricular internal dimension and respiratory variation in chamber size, with increased right ventricular internal dimension during inspiration (4,5). These findings are the echocardiographic correlates of paradoxical

pulse. Abnormal posterior motion of the anterior right ventricular wall during diastole has been reported (6).

Cardiac tamponade associated with loculated pericardial hematoma. As demonstrated by these three cases, the syndrome of cardiac tamponade can occur in the absence of pericardial effusion that envelops the heart. Cardiac tamponade may occur when external compression interferes with the diastolic filling of the heart. Localized compression, as represented by a loculated effusion, is more likely to adversely affect the filling of the thinner-walled atria and right ventricle (7). Blunt and sharp chest trauma can produce tamponade by mediastinal compression (8,9). Loculated hematomas associated with cardiac compression after cardiac surgery have been reported (10,11). The presence of pericardial adhesions is responsible for the localized nature of the hematoma in this setting. The result may be the compression of the heart in areas that are not detectable during the routine M-mode echocardiographic examination. The right atrium is frequently difficult to delineate with M-mode echocardiography, but is readily visualized by two-dimensional echocardiography, especially using the apical four chamber view (12). The right atrium may also be examined by subxiphoid and short-axis views. The use of multiple tomographic planes will maximize the ability to identify a loculated pericardial effusion.

Implications. We have demonstrated that the absence of pericardial effusion on M-mode echocardiography does not rule out life-threatening cardiac tamponade. Cardiac tamponade remains a clinical and not an echocardiographic diagnosis. Two-dimensional echocardiography, especially the four chamber view, is helpful in evaluating areas that

are not usually visualized by M-mode echocardiography and is recommended in those patients in whom the clinical picture does not correlate with the M-mode findings.

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